1. Be broad and capture forms of vascular- or cerebrovascular-related damage that are likely to contribute to cognitive impairment or dementia.

2. Address shortcomings in both mild and severe forms of vascular cognitive impairment and means to assess the transition of patients from one stage to another.

3. Recognise the importance of people who are at risk of VCI, however, their consideration under this construct should be contingent upon some level of impairment.

4. Acknowledge that the classification of a patient with a mild form of VCI (i.e. non-dementia) is not necessarily predictive of progression of the impairment to a more severe form of VCI (i.e. dementia).

5. Acknowledge that the classification of a patient with a mild form of VCI (i.e. non-dementia) is not necessarily predictive of an eventual sub-type of dementia.

Box 1: VICCCS consensus guiding principles of the refinement of the concept of VCI.

Mechanisms of cause suggested by VICCCS participants	Percentage support
Cerebral amyloid angiopathy	***93%***
Mixed forms; any neurodegenerative diseases with CVD (e.g., DLB with CVD)	***93%***
White matter hyperintensities	***93%***
Microbleeds/microhaemorrhages	***89%***
Microinfarcts	***89%***
Arteritis/vasculitis, including both local and systemic inflammatory syndromes	***82%***
Subdural or subarachnoid haemorrhage	***70%***
Option "others" for future developments	***67%***
Venous thromboses/infarcts	63%
Infectious vasculitis	53%
Hippocampal sclerosis	42%
Angiomatous lesions/vascular tumors with local steal phenomenon	33%
Chronic migraine	9%

Table 1: Clarification of the possible mechanisms of cause of either sporadic or hereditary VCI. Participants were given the opportunity to propose additional causative mechanisms to those previously listed in the *O'Brien concept* [13]. Percentage support from respondents in the final round is detailed. Those highlighted in bold italics reached consensus support of 67% and therefore are recommended.

Subtypes in the VICCCS	Descriptive terms in the VICCCS	*O'Brien concept classification and causes of sporadic VCI*
Post stroke dementia		Post stroke dementia
		Vascular dementia
Multi-infarct (cortical)		Multi-infarct dementia (cortical vascular dementia)
Subcortical ischaemic		Subcortical ischaemic vascular dementia
	Strategic infarct	Strategic-infarct dementia
	Hypoperfusion	Hypoperfusion dementia
	Haemorrhagic	Haemorrhagic dementia
	Specific arteriopathies†	Dementia caused by specific arteriopathies
Mixed dementias*		Mixed AD and vascular dementia
Mild VCI		Vascular mild cognitive impairment
	Vasculitis‡	

Table 2: VICCCS recommended subtypes and descriptive terms. Mild VCI, Post stroke dementia, Multi-infarct (cortical), Subcortical ischaemic and Mixed dementias are agreed subtypes in the VICCCS. Those in bold italics fall under the umbrella term Major VCI (VaD). Agreed revised definitions of subtypes are detailed in Box 2. *A revised holistic subtype of "Mixed dementias" was developed and agreed over the course of number of rounds to replace "Mixed AD and VaD". VICCCS agreed descriptive terms include; strategic infarct, hypoperfusion, haemorrhagic and specific arteriopathies. †Specific arteriopathies was agreed in a separate question over two rounds to include; genetic, hereditary and developmental anomalies (e.g. Fabry's disease, sickle cell disease, CADASIL, CARASIL), small vessel disease from chronic hypertension and/or diabetes, ‡inflammatory/immunological vasculitis, Moyamoya disease, and intracranial atherosclerosis.

‡Vasculitis, which was not originally part of the *O'Brien concept,* was also discussed in more detail as being an important descriptive term. The original *O'Brien concept classification and causes of sporadic vascular cognitive impairment*[13] are listed for comparison.

"Post-stroke dementia"

A patient described as having post-stroke dementia (PSD) may or may not have presented evidence of mild cognitive impairment prior to stroke. The patient may exhibit immediate AND/OR delayed cognitive decline that begins after, but within 6 months, of stroke, that does not recover. PSD results from different vascular causes and changes in brain. It includes cases with multiple corticosubcortical infarcts, strategic infarcts, subcortical ischaemic vascular dementia and various forms of neurodegenerative pathology, including AD, which develop within 6 months of stroke*. This temporal basis for cognitive decline after stroke differentiates PSD from other forms of major VCI (VaD).

"Mixed dementias"

A standalone umbrella subgroup termed "mixed dementias" includes all the phenotypes specified for each combination, i.e. VCI-AD, VCI-DLB etc. It is recommended that a patient is referred to as having "VCI-AD", according to the phenotypes present, rather than less specific "mixed dementia", for example. **Where discrimination is possible, the order of terms should reflect the relative contribution of the underlying pathology, i.e. AD-VCI, or VCI-AD.*

"Subcortical ischaemic vascular dementia (SIVaD)†*"*

Small-vessel disease is the main vascular cause of subcortical ischaemic vascular dementia. Lacunar infarct and ischaemic white-matter lesions are the main type of brain lesions, which are primarily located subcortically. It incorporates the overlapping clinical entities of Binswanger's disease and the lacunar state ‡.

"Multi-infarct dementia"

"Multi-infarct dementia (MID) relates to the involvement, and likely contribution, of multiple large cortical infarcts in the development of dementia" §.

BOX 2: VICCCS proposed definitions of Major VCI (VaD) subtypes. The above VICCCS definition of PSD is built upon the definition of O'Brien and colleagues[13].

*Since a key facet of the definition of PSD is a time component of the appearance of decline within 6 months of having a stroke that does not recover, then irrespective of the presence or absence or co-morbid neurodegenerative pathology, the aspect of time should be the primary variable for delineating between

PSD (with or without neurodegenerative pathology which if present should be described) and mixed pathology (where the contributing components are described). In other words PSD and Mixed Dementias could both have mixed pathology but PSD is recognised by its more acute presentation.

†As part of the efforts in VICCCS to standardise the nomenclature and abbreviations to be used in the future, VICCCS *diagnosis* participants were asked which abbreviation, from those most commonly used for subcortical ischaemic vascular dementia, should be taken forward. Initially no consensus was reached (SIVaD 36%; SIVD 23%; SiVaD 19%; SiVD 4%, with 18% stating no preference) but in the subsequent round, where participants were asked to choose their preference from the two most favoured abbreviations from Round 4, most support was for SIVaD (65%) and therefore adopted.

‡99% of respondents asked about this definition in the VICCCS *diagnosis* study supported this original definition[11] of subcortical ischaemic vascular dementia whilst 92% supported it as a diagnostic category. 76% of respondents stated they would use this term clinically.

§69% of VICCCS *diagnosis* respondents agreed that MID should be a diagnostic category, however opinion was split on the use of this term in the clinical setting, with only 52% in favour of it. There was a consensus (72%) support for the original definition of MID by Hachinski *et al*[9] MID reflects the traditional view that multiple large cortical infarcts are required for dementia to develop", however the most frequent objection was use of the word “required”. Therefore, to give opportunity for this objection to be considered, a modified definition was also presented along with the original definition for participants to state their support of in the subsequent round. The modified definition proposed received a consensus support (72%) is that given.

- Evidence based studies to support further sub-division of Mild VCI.
- Develop a more systematic step-wise approach towards sub-typing of patients based on new VICCCS proposed categories of Location, Aetiology, Domains (affected) and Severity.
- Investigation of factors that determine immediate or delayed onset of VCI in PSD patients and/or
- Investigation of factors (e.g. time to onset, biomarkers, cognitive parameters) that may better delineate co-morbidity of PSD with other causes of VCI or non-vascular dementias
- Further elucidation to improve phenotyping of relative contribution of the co-occurring pathology, e.g. AD-VCI, or VCI-AD in mixed dementias; or other neurodegenerative diseases (e.g. Parkinson's disease) or psychiatric disorders that co-present with CVD.
- Further exploration of the utility and validity of the traditional term multi-infarct dementia as either a specific-subtype of Major VCI (VaD) or as a complementary descriptive term alongside the newly proposed sub-types of Major VCI (VaD)

Box 3: Potential areas for future research as proposed directly or identified from responses from the VICCCS.